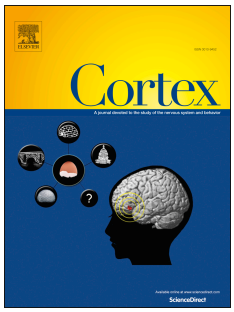
Accepted Manuscript



Altered neural dynamics in people who report spontaneous out of body experiences

Elizabeth Milne, Stephanie Dunn, Chen Zhao, Myles Jones

PII: S0010-9452(18)30354-X

DOI: https://doi.org/10.1016/j.cortex.2018.10.019

Reference: CORTEX 2453

To appear in: *Cortex*

Received Date: 17 May 2018

Revised Date: 22 September 2018

Accepted Date: 23 October 2018

Please cite this article as: Milne E, Dunn S, Zhao C, Jones M, Altered neural dynamics in people who report spontaneous out of body experiences, *CORTEX*, https://doi.org/10.1016/j.cortex.2018.10.019.

Altered neural dynamics in people who report spontaneous out of body experiences.

Elizabeth Milne*, Stephanie Dunn, Chen Zhao and Myles Jones

Department of Psychology, The University of Sheffield, Sheffield, Cathedral Court, 1 Vicar Lane, Sheffield S1 2LT, UK

*Corresponding author. Sheffield Autism Research Lab, Department of Psychology, The University of Sheffield, Cathedral Court, 1 Vicar Lane, Sheffield S1 2LT, UK

e-mail: e.milne@sheffield.ac.uk (Elizabeth Milne)

Abstract

It has been suggested that individual differences in cortical excitability leading to disruption of the timing and integration of sensory information processing may explain why some people have out of body experiences (OBE) in the absence of any known pathological or psychiatric condition. Here we recorded EEG from people who either had, or had not experienced an OBE in order to investigate the neural dynamics of OBE in the non-clinical population. A screening questionnaire was completed by 551 people, 24% of whom reported having at least one OBE. Participants who were free of any psychiatric or neurological diagnoses, including migraines, were invited to take part in subsequent EEG recording. EEG data were obtained from 19 people who had had an OBE and 20 who had not. Amplitude of the visual P1 ERP deflection and consistency of alpha-band phase locking were significantly reduced in the participants who had had an OBE. We did not find any group differences in resting state power or in visually induced gamma oscillations. These results provide support for the claim that cortical differences, particularly with respect to the timing of visual information processing, may give rise to OBE in clinically healthy individuals. To our knowledge, this study is the first to compare EEG variables obtained from people who have, and have not, had an OBE.

1. Introduction

Out-of-body experiences (OBE) are hallucinatory states during which people experience their centre of awareness as located outside of their physical body. Blanke and Arzy (2005) describe three phenomenological characteristics of OBE: disembodiment; the impression of seeing the world from a distant and elevated visuo-spatial perspective; and the experience of seeing one's own body from this elevated visual perspective. OBE have been associated with vestibular dysfunction (Lopez & Elzière, 2017) and neurological conditions such as epilepsy and seizures (Devinsky, Feldmann, Burrowes & Bromfield, 1989). OBE can be induced by stimulation of the temporoparietal junction (TPJ, Bos, Spoor, Smits, Schouten, & Vincent, 2016), and as such, one cause of OBE is considered to be a failure of multisensory integrative processes at the TPJ (Blanke & Arzy, 2005; Blanke et al., 2005).

A number of studies have reported that OBE also occur in the absence of clear medical, pharmacological or psychological correlates (Alvarado, 2000). The incidence of spontaneous OBE in the general population appears to be approximately 10%. For example, from a selection of 321 individuals on the electoral register, 12% of respondents identified that they had experienced at least one OBE (Blackmore, 1984). Studies that have recruited clinically healthy University students as participants have reported OBE occurrence in 26% (Braithwaite, Samson, Apperly, Broglia, & Hulleman, 2011), 22% (Braithwaite, Broglia, Bagshaw, & Wilkins, 2013) and 10% of the samples (Braithwaite et al., 2013). In a more recent study, occurrence of OBE was assessed in patients who had been referred to an ontoneurological clinic for assessment of dizziness and vertigo, and a matched control group with no clinical symptoms composed of relatives of the patients and hospital staff. Within these samples, OBE occurred in 14% of the patients and 5% of the control group (Lopez & Elzière, 2017).

In the non-clinical population, cortical hyperexcitability has been suggested as a potential neural correlate of OBE (Braithwaite et al., 2013a). Given the interplay between neural excitation : inhibition ratio and perception, investigation of perception can be a useful method to investigate cortical excitation and inhibition in humans (see Dickinson, Jones, & Milne, 2016; Heeger, Behrmann, & Dinstein, 2017 for reviews). For example, some people experience discomfort including headache and nausea, and see perceptual distortions such

as phantom colours, shimmering and bending of lines, when viewing certain visual patterns such as high contrast black and white stripes. This phenomenon has been termed 'pattern glare' and effects are most pronounced when stimuli are presented at spatial frequencies of 3 to 4 cycles per degree (Wilkins et al., 1984). Viewing images of this nature can lead to paroxysmal epileptiform EEG activity in people with photosensitive epilepsy (Wilkins, Binnie, & Darby, 1980), and pattern glare symptoms are reported more frequently in individuals who experience migraine with aura (Huang, Cooper, Satana, Kaufman, & Cao, 2003). Both migraine and epilepsy have been associated with elevated cortical excitability (Welch, D'andrea, Tepley, Barkley, & Ramadan, 1990), supporting the suggestion that pattern glare is due to cortical hyperexcitability. Braithwaite and colleagues found that people who had an OBE reported increased pattern glare compared with people who had not (Braithwaite et al., 2013a; Braithwaite et al., 2013b), leading to the suggestion that individual differences in cortical excitability may underlie OBE in the non-clinical population (Braithwaite et al. 2013). Further support for this position comes from subsequent work which showed that in a group of clinically healthy student volunteers, participants who experienced increased pattern-glare showed increased response to cortical excitation, delivered via anodal tDCS stimulation and decreased response to cortical inhibition delivered via cathodal stimulation (Braithwaite, Mevorach, & Takahashi, 2015).

In addition to experiencing pattern glare, the participants in Braithwaite's studies who had OBE, and / or experienced increased pattern glare, reported increased experience of other types of anomalous, hallucinatory perception, as measured with the Cardiff Anomalous Perception Scale (CAPS). The CAPS is a 32-item self-report questionnaire which asks participants whether or not they have experienced a range of anomalous perceptions, such as "Do you ever have the sensation that your limbs might not be your own or might not be properly connected to your body?" and "Do you ever feel that someone is touching you, but when you look nobody is there?" Significantly more of these items were endorsed by people who had experienced an OBE than people who had not (Braithwaite et al., 2013a) suggesting that the neural substrate underlying propensity to OBE in the non-clinical population may give rise to a variety of anomalous perceptual experiences.

There are a number of different ways that cortical hyperexcitability could manifest in individuals with OBE. Drawing on fMRI data showing that migraineurs with aura show

elevated BOLD response in the visual cortex when viewing stimuli that elicit pattern glare compared to non-migraineurs, Huang et al. (2003) have suggested that migraineurs with aura experience an imbalance in the excitatory neurotransmitter glutamate and the inhibitory neurotransmitter GABA (Huang et al., 2003). This neuronal imbalance may cause neurons to fire inappropriately leading to aura and visual distortions such as pattern glare (c.f. Braithwaite et al., 2013a; Braithwaite et al., 2013b). Additionally, disruption to the timing of visual information processing and of the integration of visual information with higher level processes could lead to an OBE. As Braithwaite et al., (2013b) point out: "The coherent sense of embodiment rests on a coordinated set of inputs from various sensory regions of the brain. If certain information is not available for integration at the correct time, or it is available but degraded in some way, then the integrative processes will seek to combine whatever compromised information is available- possibly resulting in a form of illusory conjunction …" (Braithwaite et al., 2013b, pp 553).

The aim of the current study is to use EEG to further understand the neural dynamics of people who experience OBE. The EEG signal recorded at the scalp reflects a finely tuned balance between neural excitation and inhibition (Bruyns-Haylett et al., 2017), and enables measurement of stimulus-related perturbations in neural activity with sub-millisecond precision, as such, the technique of EEG is very well suited to test the hypotheses that variation in cortical excitability, and / or disruption in the timing of visual information processing may be associated with OBE in the non-clinical population.

Following presentation of a visual stimulus, the ERP waveform takes on a characteristic appearance with identifiable peaks and troughs known as the P1-N1 complex. Increases in both EEG power and phase-locking are also seen, particularly in the alpha frequency band (8 – 13 Hz). Phase-locking can be quantified by computing inter-trial coherence (ITC). This variable, which is measured between 0 and 1, reflects the consistency of phase-angles across trials at any given time point in a time-series. Within the EEG signal, high ITC indicates consistent phase locking and has been considered to reflect stable neural networks, whereas lower ITC indicates reduced consistency of phase-locking and more noisy and unstable networks (see David et al., 2016). Reduced phase locking is likely to exert a functional effect on perception and cognition and may give rise to timing errors of the type

described by Braithwaite et al. (2013b). One aim of this study is therefore to test the hypothesis that people who have had a spontaneous OBE will have lower ITC than people who have not had a spontaneous OBE.

In addition to alpha phase-locking, a number of other EEG variables can provide insight into the integrity of neural networks underlying visual information processing. For example, visual alpha-band oscillations are considered to play a functional role in controlling the timing of information processing. According to one influential theory (Klimesch, Sauseng, & Hanslmayr, 2007), the P1 component of the visual evoked potential represents the inhibitory-phase of the alpha-oscillation which acts as an inhibitory filter, increasing signal to noise ratio during stimulus encoding and facilitating top-down integrative processes. Measuring P1 amplitude in people who have and have not had an OBE will provide insight into the emergence of networks combining bottom-up and top-down influences (Hanslmayr, Sauseng, Doppelmayr, Schabus, & Klimesch, 2005; Klimesch et al., 2007). Furthermore, presentation of a visual stimulus is associated with an increased in the power of higher frequency, gamma-band, oscillations, i.e. between 30 and 90 Hz (Schadow et al., 2007). Gamma-band oscillations are generated by the activity of inhibitory GABAergic interneurons in neuronal networks involving excitatory pyramidal cells and inhibitory interneurons (Bartos, Vida, & Jonas, 2007; Traub et al., 1998; Whittington & Traub, 2003). The peak frequency of visually induced gamma-oscillations has been shown to be related to levels of the inhibitory neurotransmitter GABA in the visual cortex (Muthukumaraswamy, Edden, Jones, Swettenham, & Singh, 2009; Cousijn et al., 2014). Specifically, higher peak frequency of visually induced gamma oscillations is associated with elevated resting occipital GABA levels. Differences in the frequency of the visual induced gamma-band response between people who have and have not had an OBE may therefore be indicative of an association between OBE and variation in the balance of neural excitation and inhibition.

Here we recorded EEG during visual stimulation from people who either have, or have not, had a spontaneous OBE. We computed three main variables of interest: P1 ERP amplitude; the consistency of alpha phase-locking following stimulus presentation (ITC); and the peak frequency of induced gamma oscillations. We hypothesised that people who have had a spontaneous OBE would show reduced P1 ERP amplitude, reduced alpha-band inter-trial

coherence, and lower peak induced gamma-frequency, than those who have not had a spontaneous OBE. We also computed power spectral density obtained from eye-closed resting data in order to compare baseline power between groups. Participants were recruited on the basis of whether or not they had ever had an OBE, and were free of any neurological, neurodevelopmental or psychiatric diagnoses.

2. Method

2.1 Participants

Participants were recruited from the student and staff body at the University of Sheffield in the UK and via social media. 551 participants completed an online screening questionnaire hosted by Qualtrics (www.qualtrics.com), which consisted of the questions presented in table 1. From this screening questionnaire, participants who met the inclusion and none of the exclusion criteria were invited to an EEG session based on whether or not they had had an OBE. Inclusion criteria for the OBE group were: participant had experienced one or more OBE, defined by answering 'yes' to Q1 of the screening questionnaire and confirmed during the lab session; and participant was over 18. Exclusion criteria for the OBE group were: history of migraines, epilepsy or seizures; a diagnosis of bipolar disorder, psychosis or schizophrenia; diagnosis of a neurodevelopmental condition; currently taking medication which may affect neurotransmitter balance (e.g. medication for anxiety / depression, or opiate based medication); reporting that the OBE occurred whilst under the influence of drugs or alcohol; or providing a description of an OBE that occurred during sleep, which is indicative of a lucid dream. Inclusion criteria for the control group were: responding 'no' to question 1 of the screening questionnaire, and being the same gender and within +/- 2 years of an OBE participant's age. Exclusion criteria for the control group were: medical history of migraines, epilepsy or seizures; diagnosis of a neurodevelopmental condition; diagnosis of bipolar disorder, psychosis or schizophrenia; or currently taking medication which may affect neurotransmitter balance.

Table 1 - OBE screening questionnaire

	Question	Response Options
1	Have you ever had an experience where you have perceived / experienced the world from a vantage point outside of the physical body? If NO, please skip to Q7. If YES:	Yes/No/DK
1a	Did you see a representation of your physical body?	Yes/No/DK
1b	Did you see the world from a perspective outside of your physical body?	Yes/No/DK
1c	Did you feel detached from the body?	Yes/No/DK
1d	Did you feel in control of your experience(s)?	Yes/No/DK
1e	How long do you estimate the experience(s) lasted?	Scale of 1 (≤1) – (≥ 30 min)
2	How frequently do the experiences occur?	1 (once) - 5 (very frequently)
3*	Did the experience occur while you were asleep?	Yes/No/DK
4*	Do you think the experience occurred as a result of drinking alcohol or from medication (either prescription or recreational)?	Yes/No/DK
5	When you have had these experiences, have they been associated with any particular time of your life?	Free response
6	Please describe these experiences in as much detail as you can. If you have had many, please describe the most common type.	Free response

7*	Do you experience any of the following conditions: migraine, epilepsy, seizure?	Yes/No/DK
8*	Do you have a diagnosis of any neurodevelopmental condition? (e.g. autism, ADHD)	Yes/No/DK, please provide detail
9*	Do you have a diagnosis of any mental health condition? (e.g schizophrenia, bipolar disorder)	Yes/No/DK, please provide detail
10	Do you experience frequent episodes of dizziness?	Yes/No/DK, please provide detail
11	Do any of your first-degree relatives have a diagnosis of any neurodevelopmental condition? (e.g. autism, ADHD)	Yes/No/DK, please provide detail
12	Do any of your first-degree relatives have a diagnosis of any mental health condition? (e.g. schizophrenia, bipolar disorder)	Yes/No/DK, please provide detail

* = exclusion criteria

All participants who completed the screening questionnaire were entered into a prize draw to win a £50 Amazon voucher. Each participant who came into the lab for the testing session was given £5 as a gesture of our thanks for their time. The study received full ethical approval from the local research ethics committee. Participants provided informed written consent, in accordance with the declaration of Helsinki.

A total of thirty-nine people took part in the EEG study, 19 participants who reported a spontaneous OBE and twenty participants who had never had an OBE. The OBE group (14 females, 5 males) had a mean age of 26.8 years (SD = 10.2) the control group (14 females, 6 males) had a mean age of 26.5 years (SD = 9.7), the mean age of the two groups was not significantly different, Mann-Whitney U = 182, p = .832, r = .042.

2.2 Measures & Procedure

2.2.1 Questionnaires

During the lab-based session the experimenter asked participants to describe their previous OBE (where applicable) to ensure that the description matched the inclusion criteria and did not fulfil any of the exclusion criteria. Participants completed the Cardiff Anomalous Perceptions Scale (CAPS, Bell, Halligan, & Ellis, 2005). Participants responded to each question with Yes/No (coded 1/0 respectively). The minimum possible score was 0 and the maximum possible score was 32. Participants also completed the Autism Spectrum Quotient (AQ, Baron-Cohen, Wheelwright, Skinner, Martin, & Clubley, 2001), which is a 50 item questionnaire that measures the degree to which adults report traits associated with the autistic phenotype. Each of the 50 items is scored as 0 or 1, with 1 being indicative of a response associated with an autistic-like trait. The AQ was administered as we have previously found that autism is associated with reduced alpha-band ITC (Milne, 2011) and elevated induced gamma-band frequency (Dickinson, Bruyns-Haylett, Smith, Jones, & Milne, 2016), and that autistic adults obtain higher CAPS scores than neurotypical controls (Milne, Dickinson, & Smith, 2017). We therefore included the AQ so that autistic traits could be accounted for by analyses of covariance if necessary. After completion of the questionnaires, EEG was recorded.

2.2.2 EEG Task

Participants were seated inside an electrically shielded chamber and asked to sit still and watch the computer screen. Stimuli were generated by the Psychtoolbox set of functions (Brainard & Vision, 1997) and presented on a 20-inch LCD screen within Matlab (The Mathworks inc.). Stimuli consisted of a black and white checkerboard which subtended 13.5 x 11.5 degrees of visual angle, presented at a distance of 57cm. Each check subtended 0.4 degrees of visual angle. Participants were asked to maintain fixation on a small red cross that appeared in the centre of the checkerboard and remained present during the inter-stimulus interval. Each checkerboard remained on screen for a mean interval of 2000 ms, jittered between 1500 and 2500 ms. The mean inter-stimulus interval was also 2000 ms, jittered between 1500 and 2500 ms. Two blocks of 100 trials were presented, the length of break between block 1 and block 2 was determined by the participant, and was typically under two minutes. Participants were instructed to press the spacebar at stimulus offset. Responses were made with the right hand in the first block and the left hand in the second block. After delivery of 200 trials, resting state data were acquired. Participants remained seated with the EEG cap in place and, when ready, were asked to close their eyes while EEG was recorded for 75 seconds.

2.3 EEG recording and pre-processing

EEG data were recorded from 64 electrodes via the BioSemi ActiveTwo system (Amsterdam, The Netherlands). Channel offsets were kept below +/-25 kΩ. EEG data were filtered online with a band-pass of 0.01-140 Hz and digitised with BioSemi ActiView software at a sampling rate of 2048 Hz. Offline processing was carried out using EEGLAB (Delorme & Makeig, 2004) and customised MATLAB (The Mathworks, Inc.) EEG data were downsampled to 512 Hz with BioSemi DBF Decimator software. Continuous data were referenced to the central channel Cz, and then high-pass filtered using a Hamming windowed finite impulse response filter with a passband edge of 1 Hz, to minimise low frequency drift. Following visual inspection, portions of data that were contaminated by gross artefacts were deleted. Channels were excluded if they showed artefacts or poor connection to the scalp in more than ten trials. The remaining data were decomposed by extended infomax ICA using the function *runica* implemented in EEGLAB. Independent components reflecting eye-movement and blinks and

clearly artifact-based components were excluded from the data (Delorme and Makeig 2004; (Zhao, Valentini, & Hu, 2015). Removed channels were then interpolated. The mean number of channels removed within each group was 2.6 (OBE) and 2 (control); the mean number of components removed within each group was 6.3 (OBE) and 6.5 (control).

Following this processing pipeline, continuous data were segmented into different files and subjected to additional processing depending on the type of analyses to be performed. Task-related data were epoched in two different ways: for ERP analysis, continuous data were filtered with a low-pass cut-off of 30Hz, and segmented into epochs with a 100 ms baseline and 600 ms post-stimulus period around the onset of stimulus presentation; for time/frequency analyses (inter-trial phase coherence and measurement of evoked and induced gamma activity), continuous data were segmented into epochs with a -1000 ms baseline and a 1500 ms post-stimulus period around the onset of the stimulus, no additional filtering was performed. The mean number of epochs retained for each group was 191 (OBE group) and 193 (control group).

Continuous data files recorded during the resting state were re-referenced to common average reference and divided into 2 second epochs. No additional filtering was performed.

2.4 Calculation of EEG variables

2.4.1 ERPs

P1 amplitude was extracted for each participant from an 'average-channel' - created by averaging the waveforms from channels P8, PO8, PO4, O2, P7, PO7, PO3 and O1 (see supplementary material). P1 amplitude was calculated by computing the mean amplitude between 80 and 130 ms on each trial and then calculating the median of these single-trial values for each participant.

In order to establish whether any observed group differences were specific to P1, N1 and P2 amplitudes were also computed. Extraction of N1 and P2 amplitudes was carried out exactly as described above, using time-windows of 120 to 170 ms (N1) and 200 to 250 ms (P2). The selection of electrodes for analysis was informed by identifying the electrodes where P1 was most prominent (see Figure 2A). Time-window selection was based on inspection of the grand-averaged ERP (see Figure 2B). Furthermore, both channel and time-window selection

were informed by, and in-line, with previous publications, e.g. see Im, Liu, Zhang, Chen, & He (2006) and Novitskiy et al., (2011).

2.4.2 Inter-Trial Coherence

The power spectrum of each trial from the average-channel described above was calculated using the *newtimef* function in EEGLAB (for a detailed description of this method see Delorme et al., 2004). The function used wavelets with 3 cycles at the lowest frequency and 12.5 cycles at the highest frequency with a window size of 556.56 ms. Spectral estimates at 200 evenly spaced time-points (from -721.5 to 1221.5 ms) and 47 evenly spaced frequencies (from 6 to 50Hz) were returned as complex vectors in phase space. After normalising the magnitude of each trial activity vector to 1, the complex average of each trial activity vector was averaged. ITC values are reported here as absolute values from these complex averages. High ITC (maximum = 1) indicates strong phase coherence, i.e., consistent alpha phase locking, whereas low ITC (minimum = 0) indicates weak phase coherence, i.e. inconsistent alpha phase locking. Time-frequency plots showing ITC at all frequencies across the epoch were plotted for each participant. Each participant showed an increase in ITC following stimulus presentation between ~50 and ~245 ms, predominantly at lower frequencies. A mean ITC value between 8 and 10 Hz and between 50 and 245 ms was computed for each participant and used as the dependent variable for subsequent group analysis.

2.4.3 Visual gamma band response

In order to avoid the contamination of artifacts such as power line interference at 50 Hz and saccadic eye movements in the visual gamma band response, time frequency analysis was applied to the timeseries from independent components (ICs) rather than from channels (Yuval-Greenberg, Tomer, Keren, Nelken, & Deouell, 2008). From each participant, the IC which showed the clearest induced gamma signal was selected for analysis. Component selection took place via the following procedure: (1) the seven ICs that accounted for the most variance to the ERP between 0 and 600 ms post stimulus onset were identified; (2) time-frequency analysis (wavelet transform) was performed on each of the identified ICs; (3) each of the resulting spectral plots was visually inspected in order to identify the component(s) which showed induced gamma power following stimulus presentation. The time frequency data associated with one IC was selected for each subject (as in Dickinson et

al., 2016b). If more than one IC within a subject showed a clear induced gamma response, the IC with the strongest (i.e. highest amplitude) power was selected. Figure 1 provides an example of a resulting time-frequency decomposition from a participant who experienced OBE, showing induced power across a large frequency range (left panel), and the same data focussed on the gamma range (middle panel). In all cases the scalp maps of each selected component showed a similar projection to that depicted in figure 1 (right panel), i.e. to the back of the head, indicating that the ICs which showed the clearest visually induced gamma response projected strongly to channels positioned above the occipital and parietal cortices. An illustrated example of the process of selecting ICs for analysis is given in supplementary material.

The timeseries of the selected ICs from each subject was analysed using wavelet transforms, replicating the procedure used in our previous work (e.g. see Dickinson et al., 2016). The complex Morlet wavelet (a complex exponential modulated by a Gaussian, ω_0= 6; where ω_0 is non-dimensional frequency) was chosen as the function ψ_0 because it provides a good balance between time and frequency localisation for feature extraction purposes (Grinsted, Moore, & Jevrejeva, 2004; Müller et al., 2004). The number of octaves per wavelet scale was set at 1/60 which provided a sufficiently 'smooth' picture of wavelet power and resulted in high spectral resolution in the gamma band range (<1Hz). Mean power for each scale during the pre-stimulus period for each trial was considered to be baseline and was subtracted from the wavelet transforms. As such, data is presented as changes in power following stimulus presentation. For measurement of induced gamma power and frequency wavelet transforms were performed on each epoch and then averaged, for measurement of evoked gamma power, the single-trial time series were averaged prior to wavelet transform.

Although our main hypothesis was that the peak frequency of induced gamma oscillations would be lower in the participants who had experienced OBE, for completeness, we also included analyses of evoked and induced power. Induced power was identified as the maximum power within the frequency band of 30 to 90 Hz. To ensure the induced signal was selected, the time window was restricted to between 200 to 1450 ms of the average of the single-trial wavelet analysis. Peak induced gamma frequency was given as the frequency of this maximum power. Evoked power was given as the mean power within the frequency

band of 30 to 80 Hz and within the time window of 50 to 200 ms of the averaged trial wavelet analysis.

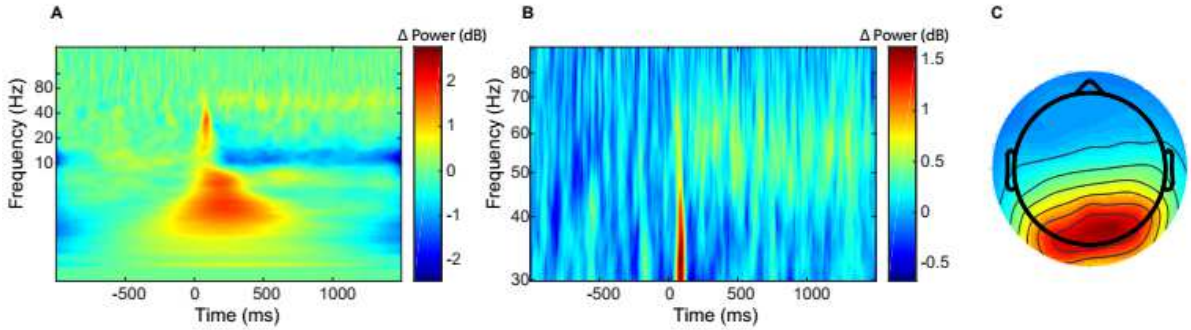


Figure 1 Time frequency decomposition and scalp map of the selected IC from a subject in the OBE group. **A** shows epoch-averaged time frequency analysis of entire frequency range. **B** shows epoch-averaged time frequency analysis of restricted frequency range (Gamma 30:90 Hz). **C** shows the scalp map of the independent component (IC) selected for time frequency analysis.

2.4.4 Power spectral density

Power spectral density value was calculated using EEGLAB function *spectopo*, which uses the *pwelch* function within Matlab (The Mathworks Inc.) to perform frequency decomposition. For analysis, channels from the following four regions were averaged: anterior left (including channels AF7, AF3, F7, F5, F3, F1, FT7, FC5, FC3, FC1, T7, C5, C3 and C1), anterior right (AF8, AF4, F8, F6, F4, F2, FT8, FC6, FC4, FC2, T8, C6, C4 and C2), posterior left (TP7, CP5, CP3, CP1, P9, P7, P5, P3, P1, PO7, PO3 and O1), and posterior right (TP8, CP6, CP4, CP2, P10, P8, P6, P4, P2, PO8, PO4, O2). Spectral power was computed from these channel-averages, and absolute alpha and gamma power were given as mean power between 8 and 13 Hz (alpha) and between 30 and 45 Hz (gamma) at each of the four regions of interest. For anlaysis, values were converted to µV^2/Hz by computing 10 ^ (spectrum / 10).

3. Results

Data were analysed using JASP (version 0.9.0.1). Parametric statistics were used to compare groups unless Shapiro-Wilks test of normality indicated that the distribution of data in either group was not normally distributed. Effect sizes for group comparisons are given by Cohen's d for normally distributed data and by rank biserial correlation (r) for non-normally distributed data. Bayes factors (BF) were also included for analyses that pertained to the main hypotheses, i.e. that P1 amplitude would be reduced in the OBE group, that ITC would be reduced in the OBE group and that induced gamma frequency would be reduced in the OBE group. We adopted the classification scheme reported by JASP, in which a BF between 1 and 3 provides only anecdotal evidence to prefer the experimental hypothesis over the null hypothesis, a BF between 3 and 10 provides moderate evidence, a BF between 10 and 30 provides strong evidence, and a BF between 30 and 100 provides very strong evidence. For all analyses we used default prior scales. For a more in depth discussion of Bayesian principles and JASP, see Marsman & Wagenmakers (2017).

3.1 OBE Screening Questionnaire

One hundred and thirty-three (24%) of the 551 participants who completed the screening questionnaire reported having had an OBE. Note that this incidence rate may be higher than would be found in the general population because those who have had an OBE may have been more likely to respond to the advertisement to take part in research investigating anomalous perception. As reported in table 2, there was no difference between people who had had an OBE and people who had not in their reports of migraines (43% and 46%), epilepsy (0% and 0.2%) or seizures (3.8% and 3.1%). However there was a significant difference in experience of dizziness between those who had and had not experienced an OBE, $\chi^2(1, N = 551) = 14.9$, $p = <.001$; a larger percentage of those who reported an OBE (35%) experienced dizziness compared to those who did not (21%). Of those who reported dizziness, 52% did not know the cause; the most common known causes were low blood pressure, anaemia, migraine, vestibular disorders and retinal disease. There was also a significant difference between those who had had an OBE and those who had not in their report of being diagnosed with a mental health condition, $\chi^2(1, N = 551) = 9.7$, $p = <.05$, with a larger percentage of those who had experienced an OBE (39%) reporting a mental health

condition compared to those who did not (25%). Of those who reported a mental health diagnosis, the most common were anxiety (66%) and depression (54%) followed by bipolar disorder (1%) and psychosis (1%).

Table 2 - A summary of the results from the OBE screening questionnaire

Question	OBE	Non-OBE	Group Difference
Gender	97 Female (72%), 35 Male, 1 Transgender	275 Female (66%), 142 Male, 1 genderfluid	$\chi^2 = 0.18$
Age (S.D.)	27.1 (9.9)	25.5 (9.9)	t = 0.12
Saw a representation of their physical body	56 (42%)	NA	
Saw the world from a perspective outside of their physical body	86 (65%)	NA	
Felt detached from the body	95 (71%)	NA	
Felt in control of experience(s)	40 (30%)	NA	
Length of experience	<1min: 42 (32%) 1-3 mins: 80 (60%) >30 mins: 11(8%)	NA	
OBE occurred during sleep	6 (5%)	NA	
OBE occurred as a result of drinking alcohol or from medication (either prescription or recreational)	3 (2%)	NA	
Migraine	57 (43%)	195 (46%)	$\chi^2 = 0.07$
Epilepsy	0	1 (0.2%)	$\chi^2 = 0.32$

Seizure	5 (4%)	13 (3%)	$\chi^2 = 0.14$
Diagnosis of a neurodevelopmental condition (e.g. autism, ADHD)	11 (8%)	47 (11%)	$\chi^2 = 0.95$
Diagnosis of a mental health condition (e.g schizophrenia, bipolar disorder)	52 (39%)	105 (25%)	$\chi^2 = 9.7$*
Experience frequent episodes of dizziness	46 (35%)	88 (21%)	$\chi^2 = 14.9$**
First-degree relatives with a diagnosis of a neurodevelopmental condition (e.g. autism, ADHD)	47 (35%)	34 (8%)	$\chi^2 = 0.35$
First-degree relatives with a diagnosis of a mental health condition (e.g. schizophrenia, bipolar disorder)	13 (9%)	13 (9%)	$\chi^2 = 0.20$

* = p<.05, ** = p<.01

The following results pertain only to the participants who came into the lab to take part in the EEG session.

3.2 CAPS and AQ score

The OBE group obtained significantly higher CAPS scores than the control group (mean = 11.68, SD = 4.8, range 0 - 22 and mean = 4.8, SD = 5.3, range 3 – 26, respectively), Mann-Whitney *U* = 48, *p* = <.001, r = .747 There was no significant group difference in AQ score t(37) = .311, *p* = .758, d = .099. The AQ score range in the control group was 7-30 (mean = 17.8, SD = 6.9) and 12-29 (mean = 18.4, SD = 5.5) in the OBE group. As no group differences in AQ score were seen, this variable was not used as a covariate in any further analyses.

3.3 EEG Paradigm

3.3.1 Accuracy and Reaction Time

Mean time to respond to stimulus offset was 395.60 (116.33) ms. There was no significant difference between response times of the participants who had (M = 426.93, SD = 135.29) and had not had an OBE (M = 365.83, SD = 88.36), Mann-Whitney U = 132, p = .107, r = .305. Furthermore, there was no significant difference in the number of responses missed by the two groups, Mann-Whitney U = 151, p = .203, r = .305. The OBE group missed 4.5 (out of 200) responses on average and the control group missed 2.5 responses.

3.3.2 ERP amplitude

Data from two participants (one from each group) were excluded from ERP analyses as they did not show a clear P1 peak within the 80 to 130 ms time-window (the typical latency of the first clear peak within the ERP of these participants was ~ 180 ms). P1 amplitude was significantly reduced in the OBE group compared to the control group, Mann-Whitney U = 242, p = .031, r = .415, BF = 4.452. In order to establish whether difference in amplitude between the two groups was specific to the P1 deflection we also compared N1 and P2 amplitudes; no group differences were found: N1: t(35) = 0.469, p = .642, d = .154, BF = 0.458, P2: t(33) = 1.083, p = .286, d = .356, BF = 0.833. ERP data are shown in Figure 2.

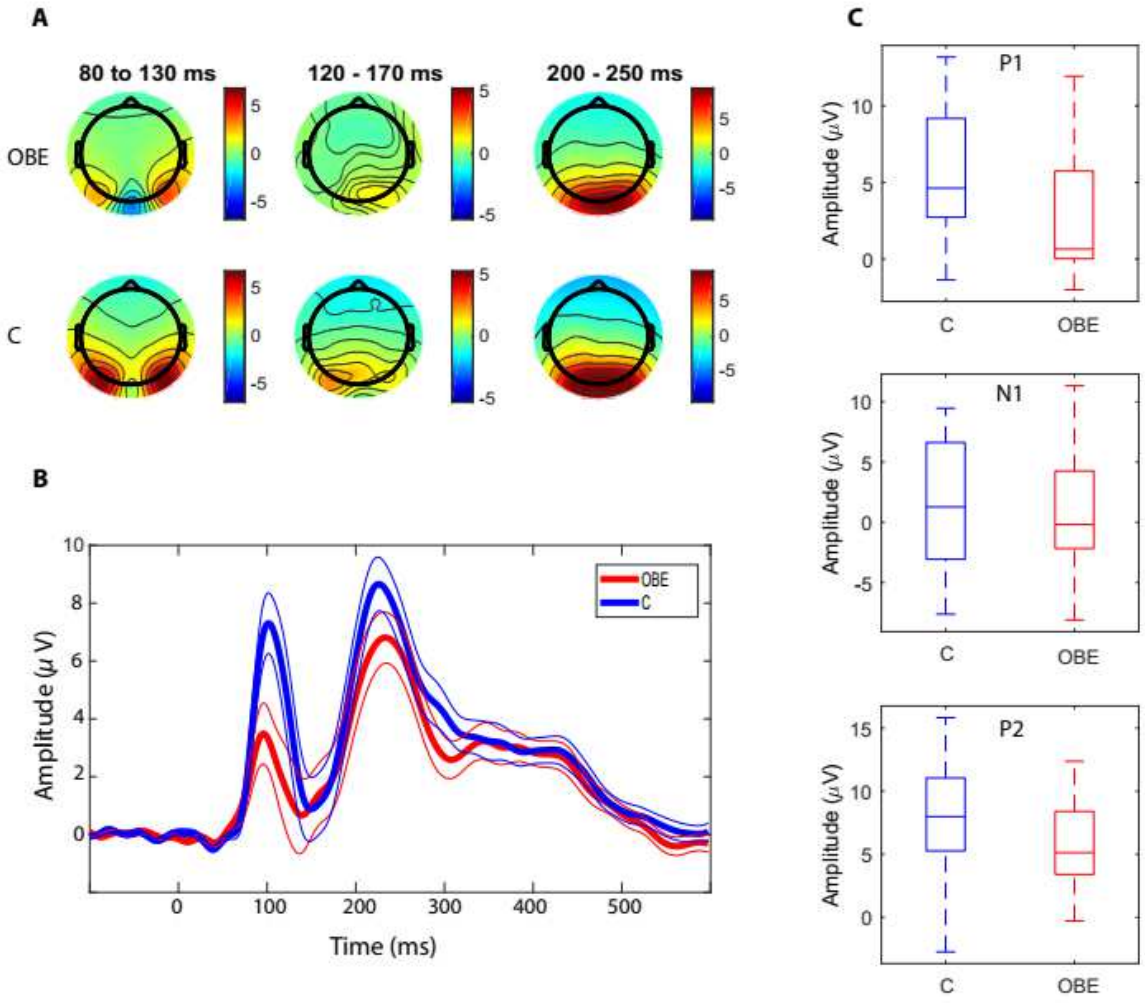


Figure 2 ERP results. **A** shows the distribution of amplitude across the scalp in the OBE group (top row) and control group (bottom row) averaged across the specified time-windows which correspond to the P1, N1 and P2 deflections. **B** shows the grand-average ERP from the channel average (see method section) in the two groups; the thick lines show the group mean and the thin lines show + / 1 one standard error around the mean. **C** shows box plots of mean amplitude for P1 (top), N1 (middle) and P2 (bottom). Note the only significant group difference was seen in P1 amplitude.

3.3.3 ITC

Time-frequency plots indicated that ITC increased in all participants following stimulus presentation, therefore data from all participants were analysed. Alpha-band ITC was significantly lower in the participants who had experienced OBE than those who had not, t(37) =2.515, p = .016, d = .806, BF = 6.738. Time-frequency plots averaged across the OBE

and control groups, alongside box-plots showing the distribution of mean ITC values are shown in figure 3.

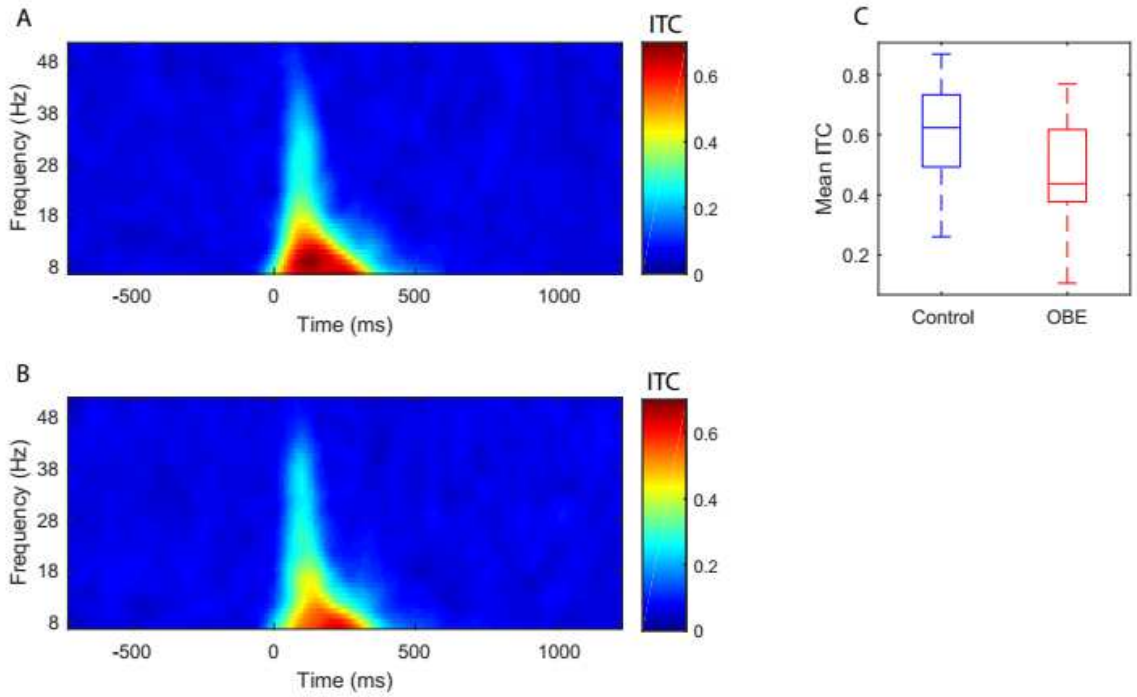


Figure 3. ITC results. **A** and **B** show average time-frequency plots of ITC in the control (**A**) and OBE groups (**B**) as computed from the channel average. The box-plot in **C** shows ITC data in both groups computed as mean ITC between 50 and 245 ms and between 8 to 10 Hz.

3.3.4 Visual Gamma Band

There was no significant difference between the OBE and control group in any of the variables calculated from the visual gamma band response. Mean (and SD) evoked gamma power in the OBE and control groups was 2.88 dB (0.66) and 2.93 dB (0.57), Mann-Whitney = 196.5, p = .866, r = .034. Mean Induced gamma power was 0.99 dB (0.39) and 1.25 dB (0.62), Mann-Whitney = 230, p = .267, r = .211. Mean peak induced gamma frequency was 57.48 Hz (16.88 Hz) and 57.88 Hz (21.65) in the OBE and control group, Mann-Whitney = 190.5, p = 1, r = .003, BF = 0.316. Time-frequency plots showing average induced power for the OBE and control group are shown in supplementary material.

3.3.5 Power spectral density

Figure 4 shows the mean power spectrum and the scalp-distribution of alpha and gamma power in the two groups. Visual inspection of this plot suggested that the two groups may differ in alpha and / or gamma power, therefore these frequency bands were investigated further. There were no statistically significant differences in alpha power between the participants who had and had not experienced an OBE at any of the four regions of interest, all $t < 1$; all $p \geq 0.519$, all $d \leq 0.209$. There was a significant difference in spontaneous gamma power between the two groups when measured over the left anterior region only. Specifically, gamma power was lower in the OBE group (mean = 0.07 dB, SD = 0.07), than in the control group (mean = 0.14 dB, SD = 0.11), $t(37) = 2.390$, $p = .022$, $d = .766$. However, when corrected for multiple comparisons, this difference was no longer significant.

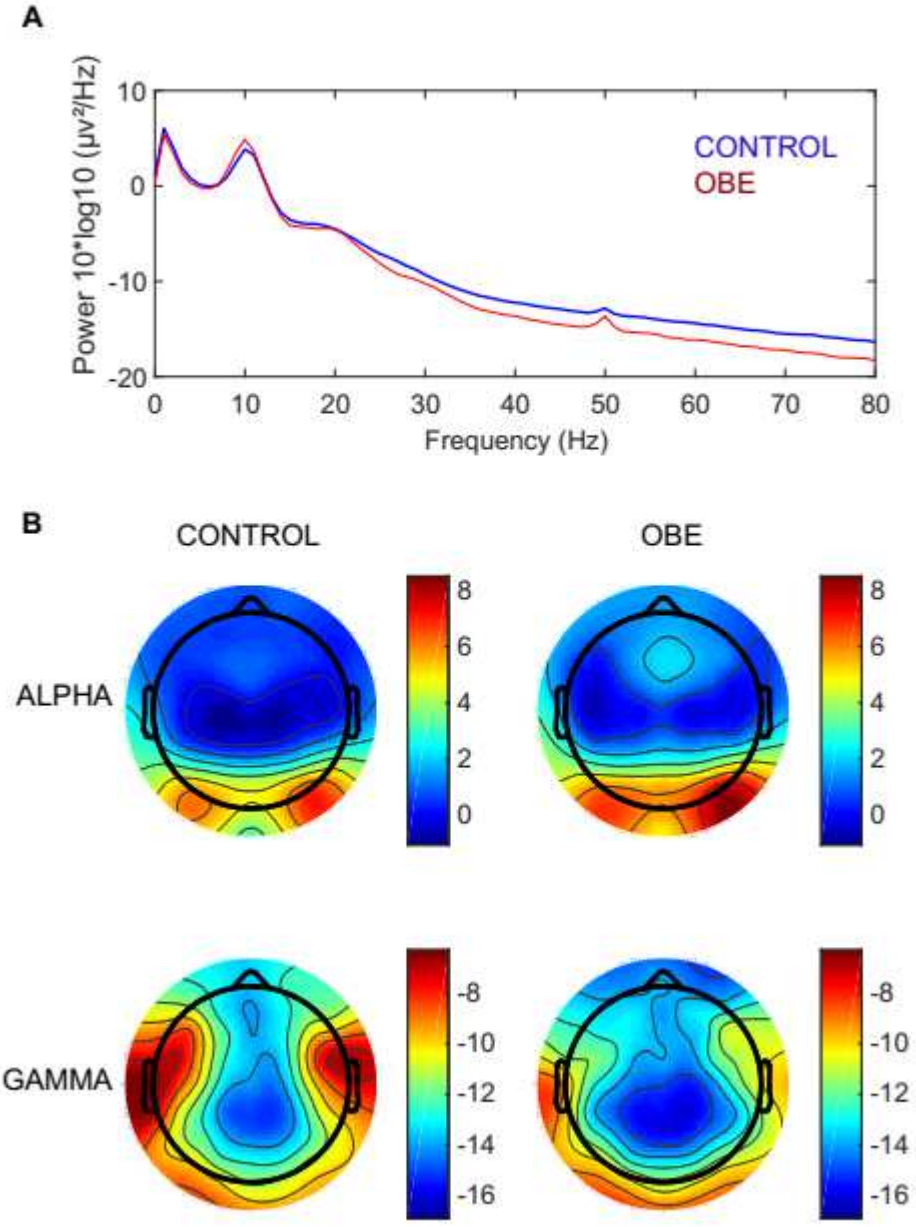


Figure 4. Spectral power during eyes-closed rest in the two groups. A shows the power spectrum of each group, calculated from the average of all channels. **B** shows scalp topographies of alpha and gamma band power in the control and OBE group.

4. Discussion

The aim of this study was to gain further insight into the potential neural correlates of spontaneous OBE that occur in the absence of any known neurological or psychiatric condition. Specifically, it has been suggested that OBE may arise from inconsistency in the timing of neural activity underlying visual perception, potentially occurring as a result of cortical hyper-excitability (Braithwaite et al., 2013b). We recruited healthy participants on the basis of whether or not they had experienced a spontaneous OBE, and compared visually-elicited EEG and eyes-closed resting-state EEG between the two groups. Participants with OBE had smaller P1 amplitude and reduced inter-trial alpha-phase coherence than participants without OBE. In line with (Braithwaite et al., 2013a) we also found increased report of anomalous perceptual experiences, i.e. increased CAPS scores, in the participants with OBE. Contrary to our prediction, we did not find any difference in induced gamma frequency between the groups.

The P1 deflection arises from increased power associated with activation of neural networks, and may, in-part, arise from the resetting of on-going alpha phase following stimulus presentation (Klimesch, Sauseng, & Gruber, 2009; Makeig et al., 2002). Sources of the P1 have been localised to the extra-striate cortex (Di Russo, Martínez, Sereno, Pitzalis, & Hillyard, 2002), and, functionally, P1 is considered to reflect the encoding and integration of bottom-up and top-down visual information. As described in the Introduction, it has been proposed that on-going alpha phase controls cortical activation, and the P1 ERP deflection reflects the deactivating phase of alpha which acts as an inhibitory filter to increase signal to noise ratio during perceptual encoding (Klimesch et al., 2009). Therefore, smaller P1 amplitude in the OBE group may indicate reduced signal to noise ratio in participants who experience OBE. The latency of the P1 (~ 100 ms) occurs at a time when bottom-up signals are modulated by top-down influences (Hillyard & Anllo-Vento, 1998). Reduced signal to noise ratio in this time period, especially when coupled with reduced alpha phase-locking could lead to inaccuracies during stimulus classification and identification, potentially giving rise to anomalous perceptual experiences such as spontaneous OBE in clinically healthy individuals. Furthermore, the predictive coding model of perception assumes that the brain generates predictions about the world by combining prior beliefs with incoming sensory signals which are then tested against sensory evidence. One can argue that OBE represents

an example of false inference about the state of the self which may reflect predicting coding errors; indeed such claims have been made in an attempt to explain some of the cognitive and perceptual symptoms of autism and psychosis (Fletcher & Frith, 2009; Van de Cruys et al., 2014). Predictive coding is also assumed to underlie the dynamic coordination of neural oscillations (Friston, Bastos, Pinotsis, & Litvak, 2015). In particular, there is evidence that neuronal signalling of predictions is mediated by alpha oscillations (Bauer, Stenner, Friston, & Dolan, 2014). Taken together with the findings reported here, it is possible that reduced P1 amplitude and reduced alpha-phase coherence therefore reflect weaker capacity for feedback signalling of predictions which results in some people being more likely to experience an OBE than others.

Further support for the claim that disruption to the timing of visual information processing, and / or to the strength of the neuronal signalling of predictions, may lead to OBE comes from existing evidence that reduced alpha phase-locking occurs in clinical conditions that are associated with anomalous perception such as schizophrenia (Shin et al., 2010) and autism spectrum conditions (Milne, 2011; Milne et al., 2017). In addition, administration of ketamine leads to a reduction of alpha ITC in mice (Saunders, Gandal, & Siegel, 2012) and is known to give rise to dissociative experiences such as OBE in humans (Wilkins, Girard, & Cheyne, 2011). Such an association between clinical symptoms and pharmacological intervention provides strong indication that reduced alpha phase-locking may give rise to OBE in the non-clinical population, as well as in clinical cases.

The groups did not differ in baseline alpha power recorded during a period of eyes-closed rest, suggesting that differences in alpha oscillations in people with OBE are associated with the processing of visual information and / or stimulus triggered phase resetting rather than with on-going spontaneous alpha oscillations. Furthermore, neither N1 nor P2 amplitude differed between the groups. Following the argument of Klimesch et al., (2009) it is the positive phase of the VEP, i.e. the P1, that represents the inhibitory phase of alpha, therefore the fact that N1 and P2 amplitude did not differ between the two groups suggests that it is the inhibitory phase of alpha that is disrupted in OBE.

Although the groups did not differ in spontaneous alpha power, there was a trend towards reduction of spontaneous gamma power from left frontal electrodes during eyes-closed rest

in the participants with OBE. Interestingly, spontaneous frontotemporal gamma power has also been associated with lucid dreaming (Voss et al., 2014). However, our results were somewhat inconclusive with respect to gamma oscillations as we found no differences in visually evoked or induced gamma power or peak induced frequency between participants who did and did not experience OBE, and the difference in left-frontal resting gamma power was not significant after correcting for multiple comparisons. The data did not support our hypothesis that participants with OBE would have lower peak gamma frequency than those without OBE suggesting that baseline levels of resting GABA concentration are not different between those who have and have not had an OBE.

Twenty-four per cent of the respondents to the screening questionnaire reported having at least one OBE. This is in-line with other studies that have used a similar approach to participant recruitment (e.g. Braithwaite et al., 2013a), but higher than studies that have used different sampling methods (e.g. Lopez and Elzière, 2017 and Blackmore, 1984). Our aim was to use the screening questionnaire to identify people who had had OBE in order to invite them to take part in the EEG study. This figure therefore cannot be taken as a prevalence estimate as we advertised the research as a study investigating unusual perceptual experience and may therefore have obtained responses from a disproportionately large number of people who are prone to OBE.

The number of participants who reported having migraines in the screening questionnaire was very high, and did not differ between those people who had had an OBE and those who had not (see table 1). A migraine incidence rate of ~44% of the sample is higher than published prevalence reports, which are typically about 14% (Burch, Loder, Loder, & Smitherman, 2015). However, we didn't provide a strict definition of migraine in the questionnaire therefore it is possible that participants over-reported their experience of migraine. Nevertheless, if any participant responded yes to this question they were excluded from the EEG study. The proportion of people who were diagnosed with a mental health condition was higher in those people who had had an OBE group than in those who had not. Furthermore, more participants in the OBE group than in the control group reported experiencing periods of dizziness. This supports previous data showing that patients who were being seen in an ontoneurological clinic for dizziness reported higher levels of OBE than matched controls (Lopez and Elzière, 2017). While all of the participants

who took part in the EEG section of this study were free of any clinical diagnosis, some participants did report experiencing episodes of dizziness. The cause of these episodes, where known, included anaemia and being deficient in vitamin B12.

Although we screened for overt clinical and / or psychiatric conditions which may be related to OBEs, the extent to which the participants who experienced OBEs showed variation in psychological correlates that have been associated with OBEs remains unknown. For example, OBEs have previously been linked to multisensory own-body processing (Blanke et al., 2005) and visuo-spatial perspective taking (see Braithwaite & Dent, 2011; Braithwaite et al., 2011); hypomanic excitement (McCreery & Claridge, 1995), and to weak synaesthesia (Terhune, 2009). Future work is required to establish to what extent these features may be related to the group differences in EEG signals reported here. Furthermore, OBE is not a unitary phenomenon. There may be multiple routes to an OBE which arise from disruption to different neural and neurocognitive mechanisms. There are also differences in the subjective experience of an OBE (see Braithwaite & Dent, 2011 for a discussion). For example, while some accounts of an OBE describe the experience as the spontaneous perception of the world from a non-physical and external viewpoint, others include descriptions of own-body perception from an external viewpoint, and describe an experience that can be induced as well as occurring spontaneously. Our selection criteria were not nuanced enough to differentiate between potential taxonomies of the OBE, however future research would benefit from a more detailed understanding of each participants' specific experiences to identify whether participants fall into particular subtypes based on the phenomenology of their experiences, and if so, whether alterations to neural dynamics are seen across all subtypes or can be linked with specific experiences.

5. Conclusion

To conclude, here we find differences in neural dynamics between healthy participants who have had an OBE and participants who have not. The clearest differences between the groups were found in the visual alpha response: P1 amplitude and inter-trial alpha phase coherence were significantly reduced in the participants with OBE compared to the control group. To the best of our knowledge, this is the first report of EEG data obtained from clinically healthy people who experience OBE. We propose that inconsistency in the timing

and control of visual information processing, mediated by alpha oscillations, represents a neural vulnerability for OBE in the non-clinical population.

Acknowledgements

We would like to thank all of the participants who gave their time to take part in this study. The research was funded by a bursary grant awarded to the first author (EM) by the BIAL foundation (#86/16).

Declarations of interest: none.

References

Alvarado, C. S. (2000). Out-of-body experiences. *Varieties of anomalous experience: Examining the scientific evidence*, 183-218.

Baron-Cohen, S., Wheelwright, S., Skinner, R., Martin, J., & Clubley, E. (2001). The autism-spectrum quotient (AQ): Evidence from asperger syndrome/high-functioning autism, malesand females, scientists and mathematicians. *Journal of autism and developmental disorders, 31*(1), 5-17.

Bartos, M., Vida, I., & Jonas, P. (2007). Synaptic mechanisms of synchronized gamma oscillations in inhibitory interneuron networks. *Nature reviews neuroscience, 8*(1), 45.

Bauer, M., Stenner, M.-P., Friston, K. J., & Dolan, R. J. (2014). Attentional modulation of alpha/beta and gamma oscillations reflect functionally distinct processes. *Journal of Neuroscience, 34*(48), 16117-16125.

Bell, V., Halligan, P. W., & Ellis, H. D. (2005). The Cardiff Anomalous Perceptions Scale (CAPS): a new validated measure of anomalous perceptual experience. *Schizophrenia bulletin, 32*(2), 366-377.

Blackmore, S. J. (1984). A psychological theory of the out-of-body experience. *The Journal of Parapsychology, 48*(3), 201.

Blanke, O., & Arzy, S. (2005). The out-of-body experience: disturbed self-processing at the temporo-parietal junction. *Neuroscientist, 11*(1), 16-24. doi: 10.1177/1073858404270885

Blanke, O., Mohr, C., Michel, C. M., Pascual-Leone, A., Brugger, P., Seeck, M., . . . Thut, G. (2005). Linking out-of-body experience and self processing to mental own-body imagery at the temporoparietal junction. *Journal of Neuroscience, 25*(3), 550-557.

Bos, E. M., Spoor, J. K., Smits, M., Schouten, J. W., & Vincent, A. J. (2016). Out-of-body experience during awake craniotomy. *World neurosurgery, 92*, 586. e589-586. e513.

Brainard, D. H., & Vision, S. (1997). The psychophysics toolbox. *Spatial vision, 10*, 433-436.

Braithwaite, J. J., Broglia, E., Bagshaw, A. P., & Wilkins, A. J. (2013). Evidence for elevated cortical hyperexcitability and its association with out-of-body experiences in the non-clinical population: new findings from a pattern-glare task. *Cortex, 49*(3), 793-805. doi: 10.1016/j.cortex.2011.11.013

Braithwaite, J. J., Broglia, E., Brincat, O., Stapley, L., Wilkins, A. J., & Takahashi, C. (2013). Signs of increased cortical hyperexcitability selectively associated with spontaneous anomalous bodily experiences in a nonclinical population. *Cognitive neuropsychiatry, 18*(6), 549-573.

Braithwaite, J. J., & Dent, K. (2011). New perspectives on perspective-taking mechanisms and the out-of-body experience. *Cortex, 47*(5), 628-632.

Braithwaite, J. J., Mevorach, C., & Takahashi, C. (2015). Stimulating the aberrant brain: Evidence for increased cortical hyperexcitability from a transcranial direct current stimulation (tDCS) study of individuals predisposed to anomalous perceptions. *Cortex, 69*, 1-13.

Braithwaite, J. J., Samson, D., Apperly, I., Broglia, E., & Hulleman, J. (2011). Cognitive correlates of the spontaneous out-of-body experience (OBE) in the psychologically normal population: evidence for an increased role of temporal-lobe instability, body-distortion processing, and impairments in own-body transformations. *Cortex, 47*(7), 839-853. doi: 10.1016/j.cortex.2010.05.002

Bruyns-Haylett, M., Luo, J., Kennerley, A. J., Harris, S., Boorman, L., Milne, E., . . . Jones, M. (2017). The neurogenesis of P1 and N1: A concurrent EEG/LFP study. *Neuroimage, 146*, 575-588.

Burch, R. C., Loder, S., Loder, E., & Smitherman, T. A. (2015). The prevalence and burden of migraine and severe headache in the United States: updated statistics from government health surveillance studies. *Headache: The Journal of Head and Face Pain, 55*(1), 21-34.

Cousijn, H., Haegens, S., Wallis, G., Near, J., Stokes, M. G., Harrison, P. J., & Nobre, A. C. (2014). Resting GABA and glutamate concentrations do not predict visual gamma frequency or amplitude. *Proceedings of the National Academy of Sciences*, 201321072.

David, N., Schneider, T. R., Peiker, I., Al-Jawahiri, R., Engel, A. K., & Milne, E. (2016). Variability of cortical oscillation patterns: A possible endophenotype in autism spectrum disorders? *Neuroscience & Biobehavioral Reviews, 71*, 590-600.

Delorme, A., & Makeig, S. (2004). EEGLAB: an open source toolbox for analysis of single-trial EEG dynamics including independent component analysis. *Journal of neuroscience methods, 134*(1), 9-21.

Di Russo, F., Martínez, A., Sereno, M. I., Pitzalis, S., & Hillyard, S. A. (2002). Cortical sources of the early components of the visual evoked potential. *Human brain mapping, 15*(2), 95-111.

Dickinson, A., Bruyns-Haylett, M., Smith, R., Jones, M., & Milne, E. (2016). Superior orientation discrimination and increased peak gamma frequency in autism spectrum conditions. *Journal of abnormal psychology, 125*(3), 412.

Dickinson, A., Jones, M., & Milne, E. (2016). Measuring neural excitation and inhibition in autism: Different approaches, different findings and different interpretations. *Brain research, 1648*, 277-289.

Fletcher, P. C., & Frith, C. D. (2009). Perceiving is believing: a Bayesian approach to explaining the positive symptoms of schizophrenia. *Nature Reviews Neuroscience, 10*(1), 48.

Friston, K. J., Bastos, A. M., Pinotsis, D., & Litvak, V. (2015). LFP and oscillations—what do they tell us? *Current opinion in neurobiology, 31*, 1-6.

Grinsted, A., Moore, J. C., & Jevrejeva, S. (2004). Application of the cross wavelet transform and wavelet coherence to geophysical time series. *Nonlinear processes in geophysics, 11*(5/6), 561-566.

Hanslmayr, S., Sauseng, P., Doppelmayr, M., Schabus, M., & Klimesch, W. (2005). Increasing individual upper alpha power by neurofeedback improves cognitive performance in human subjects. *Applied psychophysiology and biofeedback, 30*(1), 1-10.

Heeger, D. J., Behrmann, M., & Dinstein, I. (2017). Vision as a beachhead. *Biological psychiatry, 81*(10), 832-837.

Hillyard, S. A., & Anllo-Vento, L. (1998). Event-related brain potentials in the study of visual selective attention. *Proceedings of the National Academy of Sciences, 95*(3), 781-787.

Huang, J., Cooper, T. G., Satana, B., Kaufman, D. I., & Cao, Y. (2003). Visual distortion provoked by a stimulus in migraine associated with hyperneuronal activity. *Headache: The Journal of Head and Face Pain, 43*(6), 664-671.

Im, C.-H., Liu, Z., Zhang, N., Chen, W., & He, B. (2006). Functional cortical source imaging from simultaneously recorded ERP and fMRI. *Journal of neuroscience methods, 157*(1), 118-123.

Klimesch, W., Sauseng, P., & Gruber, W. (2009). The functional relevance of phase reset: a comment to Risner et al.(2009): The visual evoked potential of surface alpha rhythm phase. *Neuroimage, 47*(1), 5-7.

Klimesch, W., Sauseng, P., & Hanslmayr, S. (2007). EEG alpha oscillations: the inhibition–timing hypothesis. *Brain research reviews, 53*(1), 63-88.

Lopez, C., & Elzière, M. (2017). Out-of-body experience in vestibular disorders–A prospective study of 210 patients with dizziness. *Cortex*.

Marsman, M., & Wagenmakers, E.-J. (2017). Bayesian benefits with JASP. *European Journal of Developmental Psychology, 14*(5), 545-555.

McCreery, C., & Claridge, G. (1995). Out-of-the-body experiences and personality. *Journal of the Society for Psychical Research*.

Milne, E. (2011). Increased intra-participant variability in children with autistic spectrum disorders: evidence from single-trial analysis of evoked EEG. *Frontiers in psychology, 2*, 51.

Milne, E., Dickinson, A., & Smith, R. (2017). Adults with autism spectrum conditions experience increased levels of anomalous perception. *PloS one, 12*(5), e0177804.

Müller, M., Rossbach, W., Mann, K., Röschke, J., Müller-Siecheneder, F., Blümler, M., . . . Benkert, O. (2004). Subchronic effects of olanzapine on sleep EEG in schizophrenic patients with predominantly negative symptoms. *Pharmacopsychiatry, 37*(04), 157-162.

Muthukumaraswamy, S. D., Edden, R. A., Jones, D. K., Swettenham, J. B., & Singh, K. D. (2009). Resting GABA concentration predicts peak gamma frequency and fMRI amplitude in response to visual stimulation in humans. *Proceedings of the National Academy of Sciences, 106*(20), 8356-8361.

Novitskiy, N., Ramautar, J. R., Vanderperren, K., De Vos, M., Mennes, M., Mijovic, B., . . . Lagae, L. (2011). The BOLD correlates of the visual P1 and N1 in single-trial analysis of simultaneous EEG-fMRI recordings during a spatial detection task. *Neuroimage, 54*(2), 824-835.

Saunders, J. A., Gandal, M. J., & Siegel, S. J. (2012). NMDA antagonists recreate signal-to-noise ratio and timing perturbations present in schizophrenia. *Neurobiology of disease, 46*(1), 93-100.

Schadow, J., Lenz, D., Thaerig, S., Busch, N. A., Fründ, I., Rieger, J. W., & Herrmann, C. S. (2007). Stimulus intensity affects early sensory processing: visual contrast modulates evoked gamma-band activity in human EEG. *International Journal of Psychophysiology, 66*(1), 28-36.

Shin, Y.-W., Krishnan, G., Hetrick, W. P., Brenner, C. A., Shekhar, A., Malloy, F. W., & O'donnell, B. F. (2010). Increased temporal variability of auditory event-related potentials in schizophrenia and Schizotypal Personality Disorder. *Schizophrenia research, 124*(1), 110-118.

Terhune, D. B. (2009). The incidence and determinants of visual phenomenology during out-of-body experiences. *cortex, 45*(2), 236-242.

Traub, R. D., Spruston, N., Soltesz, I., Konnerth, A., Whittington, M. A., & Jefferys, J. G. (1998). Gamma-frequency oscillations: a neuronal population phenomenon, regulated by synaptic and intrinsic cellular processes, and inducing synaptic plasticity. *Progress in neurobiology, 55*(6), 563-575.

Van de Cruys, S., Evers, K., Van der Hallen, R., Van Eylen, L., Boets, B., de-Wit, L., & Wagemans, J. (2014). Precise minds in uncertain worlds: Predictive coding in autism. *Psychological review, 121*(4), 649.

Voss, U., Holzmann, R., Hobson, A., Paulus, W., Koppehele-Gossel, J., Klimke, A., & Nitsche, M. A. (2014). Induction of self awareness in dreams through frontal low current stimulation of gamma activity. *Nature neuroscience, 17*(6), 810.

Welch, K., D'andrea, G., Tepley, N., Barkley, G., & Ramadan, N. (1990). The concept of migraine as a state of central neuronal hyperexcitability. *Neurologic clinics, 8*(4), 817-828.

Whittington, M. A., & Traub, R. D. (2003). Interneuron diversity series: inhibitory interneurons and network oscillations in vitro. *Trends in neurosciences, 26*(12), 676-682.

Wilkins, A., Binnie, C., & Darby, C. (1980). Visually-induced seizures. *Progress in Neurobiology, 15*(2), 85-117.

Wilkins, A. J., Nimmo-Smith, I., Tait, A., McManus, C., Sala, S. D., Tilley, A., . . . Scott, S. (1984). A neurological basis for visual discomfort. *Brain, 107*(4), 989-1017.

Wilkins, L. K., Girard, T. A., & Cheyne, J. A. (2011). Ketamine as a primary predictor of out-of-body experiences associated with multiple substance use. *Consciousness and cognition, 20*(3), 943-950.

Yuval-Greenberg, S., Tomer, O., Keren, A. S., Nelken, I., & Deouell, L. Y. (2008). Transient induced gamma-band response in EEG as a manifestation of miniature saccades. *Neuron, 58*(3), 429-441.

Zhao, C., Valentini, E., & Hu, L. (2015). Functional features of crossmodal mismatch responses. *Experimental brain research, 233*(2), 617-629.